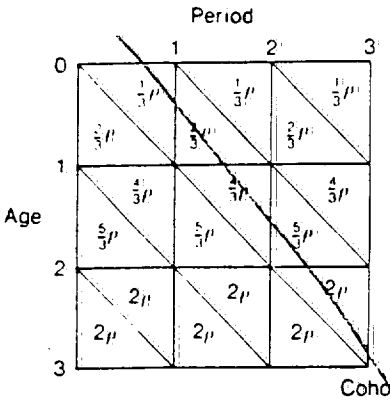

FIGURE 1. Rates for the non-linear age model, calculated in the same way as in figure 3 of Osmond and Gardner (2).

models only "work" as a result of aggregation and making assumptions of constancy of effect within an interval.

At present, we see two avenues for investigators who wish to try to estimate the separate linear effects of age, period, and cohort: 1) use a two-way table and impose a linear constraint, ignoring the overlapping of cohorts; and 2) use the individual records approach, which does not have the problem of overlapping cohorts. This approach will require a correction for potential bias brought about by the asymmetry in forcing the continuous data into a three-way table. Brown and Conelly (personal communication, 1988) have informed us of some very interesting work they are doing in this area.

Finally, in our published example on the use of individual records in the analysis of lung cancer and laryngeal cancer incidence in Scotland (3), the cohort effect is approximately quadratic and the time effect small but non-linear. Such effects cannot be induced by assuming a monotonic increasing age effect alone.

REFERENCES

1. Tango T. Re: "Statistical modelling of lung cancer and laryngeal cancer incidence in Scotland, 1960–1979." Am J Epidemiol 1988;128:677–8.
2. Osmond C, Gardner MJ. Age, period, and cohort models: non-overlapping cohorts don't resolve the identification problem. Am J Epidemiol 1989;129: 31–5.
3. Boyle P, Robertson C. Statistical modelling of lung cancer and laryngeal cancer incidence in Scotland, 1960–1979. Am J Epidemiol 1987;125: 731–44.
4. Osmond C, Gardner MJ. Age, period and cohort models applied to cancer mortality rates. Stat Med 1982;1:245–59.
5. Clayton D, Schifflers E. Models for temporal variation in cancer rates: age-period, age-cohort models. Stat Med 1987;6:449–67.
6. Fienberg SE, Mason WM. Identification and estimation of age-period-cohort in the analysis of discrete and archival data. In: Schuessler KF, ed. Sociological methodology. San Francisco: Jossey-Bass, 1978.
7. Decarli A, La Vecchia C. Age, period and cohort models: review of knowledge and implementation in GLIM. Revista di Statistica Applicata 1987; 20:397–410.
8. Holford TR. The estimation of age, period and cohort effects for vital rates. Biometrics 1983; 39:311–24.

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RE: "EFFECTS OF PASSIVE SMOKING IN THE MULTIPLE RISK FACTOR INTERVENTION TRIAL"

Some of the health effects of passive smoking may be small, and are best investigated in large cohort studies of persons exposed over a long period. It is unfortunate that the analysis by Svendsen et al. (1) of the unique data gathered in the course of the Multiple Risk Factor Intervention Trial (MRFIT) study is flawed, and may introduce confusion about the role of passive smoking as a risk factor in cardiovascular disease, and does not allow the investigators to fully explore the potential of passive smoking as a risk factor in other conditions.

The Svendsen paper repeatedly tests the statistical significance of the difference between the same proportion(s). For example, table 7 shows that of the 1,400 never smokers, 13 men died from coronary heart disease and 30 from any cause, and that there were 69 fatal or nonfatal coronary heart disease events. Each group is examined for significant difference in proportions according to the wife's smoking status as if it were independent of the two other groups; in fact, the coronary heart disease death group is a subset of the two other groups, and its contribution to the calculation of relative risk is thus taken into account three times in this table. The correct analysis would have compared "death from other causes" and "nonfatal coronary heart disease events" with "death from coronary heart disease".

The misuse of statistics is compounded in table 9, when the 2,222 ex-smokers are added to the 1,400 never smokers (this is my assumption: no *n*'s are

given). In this analysis, the 13 coronary heart disease deaths in the never smokers are again included and the proportions to which they contribute are tested for statistical significance three more times. The appropriate analysis would have examined only the 2,222 ex-smokers in the same terms, as suggested for table 7.

The reader who is interested in outcomes other than coronary heart disease death is forced to use guesswork to subtract this effect from the other data in the tables. For example, even though we are not told the numbers of men in table 9, the much lower p value for "death from any cause" than in table 7 suggests that this difference is due to the contribution of the ex-smokers. Had these been analyzed separately, the difference in risk of "death from any cause" between the exposed and nonexposed ex-smokers would probably have been even more marked. This would have suggested that the men who stopped smoking were especially susceptible to second-hand tobacco smoke. A presentation of the data that did not lump and overlap the subsets of interest would have made such speculation unnecessary.

The study by Svendsen et all is presented as an exploration without hypothesis. This "blurred" analysis could have been avoided if this report had set out to investigate an explicit hypothesis that specified the target group and the expected endpoint. Paradoxically, focussing in on a specific research question and following the method appropriate to address that question often allows the researchers to isolate and investigate secondary or unexpected results more accurately.

REFERENCE

1. Svendsen KH, Kuller LH, Martin MJ, et al. Effects of passive smoking in the Multiple Risk Factor Intervention Trial. Am J Epidemiol 1987;126:783-95.

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RE: "EFFECTS OF PASSIVE SMOKING IN THE MULTIPLE RISK FACTOR INTERVENTION TRIAL"

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Svendsen et al (1) analyze data from the Multiple Risk Factor Intervention Trial (MRFIT) study and report the relative risks of various endpoint events for men who never smoked in relation to spousal smoking. They assert that their data provide "further evidence of a potential serious health risk for a large segment of the nonsmoking population" (1, p. 792). This conclusion does not appear to be supported by the data presented.

For morbidity and mortality, the relative risks are not statistically significant, except for the "all deaths" category for the group combining "never smoked" and "ex-smoker" males. Since the relative risk for "coronary heart disease deaths" was not significantly elevated for that group, the increased relative risk for "all deaths" requires some explanation before the statistics can be assumed to indicate a meaningful increase in health risk related to spousal smoking.

While the statistics alone raise serious doubt about the conclusion of increased health risk for nonsmokers exposed to environmental tobacco smoke based on spousal smoking, questions also need to be raised about the quality of the evidence on which the assessments are based, notably the nonhomogeneity between the groups based on spousal smoking classification.

The lack of homogeneity was implicit when adjustments were made for differences in some coronary heart disease risk factors, e.g., age, weight, blood pressure, and alcohol consumption, but there is no indication that the adjustment included consideration of the additive effect of multiple risk factors, as has been demonstrated in numerous other studies, notably the Framingham Heart Study. There is no indication that other coronary heart disease risk factors, e.g., family history and exercise, were considered or adjusted for. Differences in forced expiratory volume in one second (FEV_1) among the groups were also cited. The numerous confounding coronary heart disease risk factors should not be disregarded, nor can statistical adjustments be made to eliminate their possible roles. Thus, while the MRFIT study was well designed to assess the effect of various interventions according to selected risk factors, it does not appear to have been designed to assess the environmental tobacco smoke exposure as a coronary heart disease risk factor.

Svendsen et al. observe that men whose wives smoked had "significantly lower levels of pulmonary function at baseline" (1, p. 788). The authors fail, however, to note and to interpret the data in table 6, which shows FEV_1 levels for men whose wives smoked 20+ cigarettes/day were markedly *higher* than those of men whose wives smoked 1–19 cigarettes/day, both at baseline and averaged over all visits. With such a notable reversal of the dose-response relation, which must be demonstrated if causal inferences are to be supported, there seems to be little basis for suggesting the possibility of any relation between pulmonary function and spousal smoking from this study.

The weakness of the evidence thus raises important questions about the conclusion that "passive smoking is associated with an increase in morbidity and mortality among nonsmokers" (1, p. 791). There is certainly no convincing demonstration that spousal smoking constitutes a "potential serious health risk" for any segment of the nonsmoking population.

REFERENCE

1. Svendsen KH, Kuller LH, Martin MJ, et al. Effects of passive smoking in the Multiple Risk Factor Intervention Trial. Am J Epidemiol 1987;126:783-95.

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